THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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PLEASE NOTE: Copy for illustrations for the Reporter should be sent unmounted (See PDR 36(5): 214. May 15).

WINTER INJURY TO PEACH TREES ON YUNNAN STOCK

John H. Weinberger

The need for a nematode-resistant stock for peach trees has led to the limited commercial use of seedlings of peaches introduced from Yunnan Province, China, by the Division of Plant Exploration and Introduction. These stocks have been proved to be resistant to the ordinary root-knot nematode, Meloidogyne incognita, but susceptible to another strain, M. javanica[1]. The susceptibility of Yunnan stock to some strains of nematodes and the extra cost of such stock have delayed the general adoption of the Yunnan stock in the Southeast. There is considerable interest in the use of this stock, however.

A comparison of Yunnan and Lovell stocks was started at the U. S. Horticultural Field Laboratory, Fort Valley, Georgia, in the spring of 1948. Fifteen trees of each of six varieties on Yunnan (PI 55886) and 15 of each on Lovell stock were obtained from commercial nurseries. These varieties were Dixigem, Dixired, Erly-Red-Fre, Southland, Sullivan Elberta, and FV 5-62. Two trees of each variety, one tree on Yunnan and one on Lovell seedlings, were planted in succession in rows; the rows were 18 feet apart and the trees were 12 feet apart in the row. The soil was Greenville sandy loam. Nematodes were present, but not sufficiently prevalent to noticeably affect the growth of the susceptible Lovell stocks. M. javanica was not present, for roots of the Yunnan stock were free of galls.

The trees grew moderately well, with no evidence of the incompatibility of Dixired, Southland, and FV 5-62 trees on Yunnan stock anticipated from the dwarfing observed by Bregger for certain varieties on Yunnan stock in the nursery[2]. Trees of all varieties on the Lovell stock averaged 104 mm. in trunk circumference at the end of the second growing season, and trees on Yunnan averaged 105 mm. in circumference. Only trees of the Southland variety on Lovell stocks were larger than on Yunnan.

In the spring of 1951 considerable winter injury was noted in this block of trees. Examination showed that all 14 of the injured trees were on Yunnan stock (Table 1). No trees on Lovell stock were killed. Phony disease and other causes had resulted in the earlier loss of some trees on both stocks. The winter killing was not confined to one variety, but all six varieties lost at least one tree.

FIGURE 1. Comparative winter injury to Sullivan Elberta trees on ordinary stock, left, and on Yunnan stock, right.

In the spring of 1952, after further loss of trees due to phony disease in 1951, extremely serious winter injury was evident. Table 1 shows the number of trees in the planting that were either completely killed or more than half-killed. Forty of the 45 trees on Yunnan stock were injured in this way but only 4 of the 59 trees on Lovell stock were killed. The injury was principally to the bark of the trunk, and was worst near the ground line. No damage occurred below the groundline, and the Yunnan stocks themselves, below the bud, were not hurt. The damage was to the bark of the peach variety, and none of the six varieties escaped serious damage. The lowest temperature during the winter was 17° F. on December 16, 1951.

Summarizing the two seasons' data, we find that 91 percent of the trees of all varieties on Yunnan stock were winter-killed, compared with 7 percent of the trees on Lovell stock.

The results in Table 1 include only one (PI 55886) of the six Yunnan stocks. PI 55886 is a white-fleshed clingstone peach. An unidentified freestone Yunnan, either PI 55775, 55776 or

[1]Christie, J. R., and Leon Havis. 1948. Relative susceptibility of certain peach stocks to races of the root-knot nematode. Pl. Dis. Reptr. 32:510-514.

[2]Bregger, J. T. 1948. Peach variety incompatibilities on seedlings of a Yunnan understock. Proc. Amer. Soc. Hort. Sci. 52:141-142.

Table 1. Winter injury to peach trees on Yunnan stock in Georgia.

Variety	Stock	Original number trees	1951 Number trees	1951 Number winter-killed	1952 Number trees	1952 Number winter-killed
Dixigem	Lovell	15	14	0	10	0
	Yunnan	15	12	3	6	6
Dixired	Lovell	15	11	0	7	1
	Yunnan	15	12	2	9	9
Erly-Red-Fre	Lovell	15	11	0	10	1
	Yunnan	14	10	2	6	3
Southland	Lovell	15	12	0	9	1
	Yunnan	15	12	4	8	7
Sullivan Elberta	Lovell	15	12	0	11	0
	Yunnan	13	7	1	6	6
FV 5-62	Lovell	15	15	0	12	1
	Yunnan	15	13	2	10	9
TOTAL	Lovell		75	0	59	4
	Yunnan		66	14	45	40

55887, has been used more as a stock commercially in Georgia than PI 55886. It was possible to make comparisons of winter injury in the spring of 1952 in Dixired, Dixigem, and Sullivan Elberta orchards on this stock.

In a 3,000-tree Dixired orchard, four years old, two representative 100-tree blocks on Yunnan stock were surveyed for winter injury. Eighty-three trees in one block and 77 trees in the other were winter-killed. Adjacent rows of Dixired on ordinary rootstock showed 3, 13, and 6 dead trees for 3 groups of 100 trees each. No nematode galls were found on either stock. In a 4,000-tree Dixigem orchard, three years old, 42, 18, and 13 dead trees were counted in 3 groups of 200 trees each on Yunnan stock. Adjacent trees on Lovell stock had only 2 and 4 dead trees in 200-tree blocks. Observations of injury in a four-year-old Sullivan Elberta orchard of 5,000 trees (Fig. 1) showed 78 dead trees in each of two groups of 100 trees on Yunnan, while in adjacent rows similar groups of trees on ordinary stock had only 4 and 7 dead trees, respectively. In this orchard ordinary rootstocks were moderately infested with nematodes, but Yunnan stocks were entirely free of galls. In all these orchards the original injury was to the bark of the trunk above the groundline. The Yunnan rootstocks themselves were unharmed.

For the Central Georgia region at least it appears that Yunnan rootstocks increase the susceptibility of varieties to winter injury.

U. S. HORTICULTURAL FIELD LABORATORY, FORT VALLEY, GEORGIA

STRAWBERRY FUNGICIDE SCREENING TESTS

Norman L. Horn

Considerable losses have been encountered in the strawberry growing areas of Louisiana in recent years because of fruit-rotting fungi. From observations and numerous isolations it was found that Botrytis cinerea was the major fungus involved in the fruit-rot complex. This organism has been most destructive during the beginning of the fruiting season when the temperatures were cool and the atmosphere moist. In Louisiana the most profitable part of the harvest has been at the beginning of the season. A high percentage of rot at this time, therefore, means heavy losses to growers. It was the purpose of this investigation to obtain a fungicide that could be applied prior to fruiting to reduce the amount of rot during this critical period. Powell[1] indicated that Orthocide 406 and ferbam gave field control of B. cinerea.

A fungicide screening test, including 41 treatments replicated four times, was conducted at the Louisiana State University Experiment Station. Klondike plants were double-set in hill rows and divided into plots that contained about 18 plants each. Every fungicide was applied with a three-gallon Hudson sprayer at the rate of 270 gallons per acre. The amount of material per gallon varied with the fungicide used. Applications were made on January 15, 22, 29, February 5, 12, 18, and 25.

The berries were harvested 21 times during the course of the experiment. The number of rotted and non-rotted berries was recorded as well as the weights of the latter. Because of the time element involved in separating the berries, some culls as well as the marketable fruit were included in the data. Therefore, the yields were higher than expected, but all were on a relative basis.

Three of the fungicides tested were considered to give good control of B. cinerea. These were the salt of dehydroacetic acid, manufactured by the Dow Chemical Company, Crag Experimental Fungicide 5400, manufactured by the Carbide and Carbon Chemicals Company, and Orthocide 406, manufactured by the California Spray-Chemical Corporation. The data obtained from the application of these three fungicides and the control are listed in Table 1.

Table 1. Results of four treatments in the field screening test for control of Botrytis rot of strawberry in Louisiana.

Treatment	Number rotted berries	Number non-rotted berries	Weight in ounces non-rotted berries	Number plants tested	Yields in crates per acre	Percent rot
DHA-S	210	1276	312.25	73	301	14.1
5400[a]	226	1521	358.50	73	348	12.9
406[b]	222	2011	465.50	75	441	9.9
Check	445	1233	277.00	78	251	26.5

[a] Crag Experimental Fungicide 5400
[b] Orthocide 406

Orthocide 406 reduced the rots considerably and seemed to stimulate the plants somewhat. It was the best treatment of the entire group of fungicides tested. The material was used at the rate of 6 pounds per 100 gallons of water. This amount could be reduced considerably; the reason that it was used at such a high rate was for possible phytotoxic response.

About seven weeks after the first application, the fungicides had begun to lose their effectiveness. Therefore, the field was divided into eight plots (approximately 330 plants each), four of which were treated with DHA-S and four were not sprayed. Because of its non-toxicity to humans, DHA-S could be used during the picking season. The material was used at the rate of 40 grams (97% active ingredient) per gallon (this concentration was slightly toxic to the foliage). Only one application was made followed by four harvests of three-day intervals. When three replications

[1] Powell, D. 1952. The effect of early spring fungicides on Botrytis cinerea. Plant Dis. Reptr. 36:97-98.

were used in the analysis, the DHA-S treatment was significant. There was no significance with four replications. This can be explained by the fact that in replication four of the treated plots about 35 plants were so severely injured from a previous treatment that no berries were harvested from these plants.

Two methods were used in the laboratory to detect fungicidal action on the conidia of Botrytis cinerea. One was the method by which the fungicides and conidial suspensions were mixed on glass slides and the percentage germination determined after 24 hours. In the other method, the green to white berries were dipped into combined suspensions of conidia and fungicides, and the numbers of rotted fruit compared with the numbers of unrotted fruit after a period of incubation. In four tests, conidia in contact with DHA-S, Crag Experimental Fungicide 5400, or Orthocide 406 on glass slides did not germinate, whereas there was 99 percent germination in the controls. The results of the fruit dip method are given in Table 2.

Table 2. Summary of four tests by the fruit dip method of screening fungicides for control of Botrytis rot of strawberry.

Fungicide	Total number rotted berries	Total number berries tested	Percent rot
DHA-S	16	120	13.3
Crag Experimental Fungicide 5400	41	120	24.2
Orthocide 406	43	120	35.8
Inoculated controls	101	120	84.2
Uninoculated controls	88	146	60.3

The data in Table 2 indicate that the three fungicides reduced the development of the fungus. Because of the high percentage of rotted berries in the uninoculated controls, the effects of the fungicides were probably greater than indicated, since some of the berries undoubtedly were infected before they were treated. It would seem logical that the fungus could have penetrated the fruit sufficiently to induce infection, but not to a degree to cause a noticeable spot to be formed.

Summary

Orthocide 406 was the best fungicide used in a field screening test. Not only was the amount of fruit rot reduced, but plants treated with this material seemed more vigorous than the controls. A possible recommendation in Louisiana would be the use of Orthocide 406 until fruiting time, supplemented with DHA-S, if necessary, after the harvesting period has begun. Although DHA-S showed promise as a fungicide to use on strawberries as a fruit rot control measure, its expense may limit its use. This investigation was begun before Powell published his results, so that the effect of Orthocide 406 on B. cinerea has been confirmed.

The fruit dip method was a reliable aid in screening fungicides for use on strawberries. The conventional methods of testing fungicides on glass slides also was an aid but was not so reliable as the dip method.

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RELATION OF TEMPERATURE TO PERSISTENCE OF STRAWBERRY YELLOWS VIRUS COMPLEX IN STRAWBERRY APHID[1]

P. W. Miller

Prentice (2, 3, 4), Wood and Whitehead (7), Prentice and Harris (5), Prentice and Woollcombe (6), and Mellor and Fitzpatrick (1) carried on studies on the persistence of various strawberry viruses in the body of the strawberry aphid (Capitophorus fragaefolii Ckll.). With few exceptions, persistence was determined by transfer of viruliferous aphids, after various feeding periods on infected plants, to a sucession of healthy plants. All experiments were apparently carried on at room temperature or at relatively warm temperatures in a greenhouse. Mellor and Fitzpatrick (1), in an experiment on the characteristics of the non-persistent component of the strawberry yellows virus complex, reported that aphids starved for 6 or more hours after their acquisition feeding period did not transmit this component.

In the course of some of the writer's preliminary experiments on the separation of the various components of the strawberry yellows virus complex by fasting aphids for different periods, viruliferous aphids were placed in a refrigerator maintained at 40°F. It was noted that these fasted aphids lived much longer and that the viruses which they carried persisted much longer in their bodies. In the light of this, more extensive studies were initiated to determine whether one or more of the strawberry yellows virus components persist longer in the aphid's body at relatively cool than at warmer temperatures.

In these experiments, a number of viruliferous aphids which had fed for 13 days on infected (severe yellows) plants were transferred to two petri dishes containing pieces of moist filter paper; one of the petri dishes was kept at 40°F. and the other at 70°. After fast periods of 5, 23, 43,

Table 1. Relation of temperature to persistence of strawberry yellows virus complex in strawberry aphid, 1952.

Post-acquisition fast period	Incubation temperature during the fast period	Percent of inoculated plants infected[a]	Predominant virus component in infected plants[b]
Hours	°F		
0	--	100	NP
5	40°	80	NP
23	40°	70	NP
43	40°	70	NP
69	40°	60	NP
95	40°	50	NP
141	40°	30	P
5	70°	40	NP
23	70°	30	P
43	70°	10	P
69	70°	0	--
95	70°	0	--

[a] 10 plants in each group.
[b] As indicated by characteristic symptoms in Fragaria vesca (East Malling strain). NP = non-persistent component; P = persistent component.

[1]Published as Technical Paper No. 746 with the approval of the Director of the Oregon Agricultural Experiment Station and the Chief of the Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration U. S. Department of Agriculture. Contribution of the Department of Botany and Plant Pathology of the Oregon Agricultural Experiment Station and of the Division of Fruit and Nut Crops and Diseases of the Bureau of Plant Industry, Soils, and Agricultural Engineering.

69, 95, and 141 hours, respectively, 10 of the aphids kept at 40° and 10 kept at 70° were transferred, respectively, to each of 10 healthy Fragaria vesca (East Malling strain) plants where they were permitted to feed for five days. As a check, a third lot of aphids was transferred directly from an infected (severe yellows) Marshall plant to each of 10 healthy F. vesca plants (10 aphids per plant). The relation of temperature to the persistence of the yellows virus complex in the strawberry aphid is shown in Table 1.

As shown in Table 1, the components of the strawberry yellows virus complex persisted three to four days longer in the body of the strawberry aphid at relatively cool temperatures (40°F.) than at room temperature (70°). This is particularly interesting because at room temperature the so-called non-persistent component generally dies out of the body of the aphid after about six hours of fasting. Furthermore, the aphids live correspondingly longer without food at 40° than at 70°.

It is not definitely known why the viruses should live longer at cooler temperatures. It is thought, however, that the increased persistence is probably associated with a slowing up of biological reactions, enzymic and otherwise, in both the viruses and aphids.

Apart from possible reasons as to the cause of the increased virus persistence in the aphid's body at cooler temperatures, these findings have an important bearing upon the results of previous investigations of the persistence of the components in the virus complex in the aphid's body. Past investigations on this subject have apparently been carried on at room temperature. To ascertain the absolute limits of persistence of the various virus components their persistence in the aphid's body should be reinvestigated at relatively low temperatures.

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OREGON AGRICULTURAL EXPERIMENT STATION AND U. S. BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING

A TOBACCO NECROSIS VIRUS ASSOCIATED WITH STRAWBERRY PLANTS[1]

J. P. Fulton

Tobacco necrosis virus was obtained from strawberry roots in the course of studies on strawberry viruses during the past winter. The possibility of mechanical transmission of strawberry viruses from various strawberry tissues was being considered. The tobacco necrosis virus was originally obtained from a Fragaria vesca (East Malling clone) plant showing virus symptoms similar to a combination of Type 1 and 2 viruses described by Demaree and Marcus (3). It soon became evident that it could be as readily obtained from apparently healthy East Malling clone plants and that the tobacco necrosis virus was not related to any virus symptoms on Fragaria vesca plants. Tobacco necrosis virus was also readily obtained from strawberry plants (Blakemore variety) which had been grafted to plants of the East Malling clone. It was occasionally obtained from greenhouse grown strawberry plants which had not been grafted to the East Malling clone.

Although detailed studies were not made it was assumed that the virus obtained was one of the tobacco necrosis group. Pinto bean was used as a test plant and on this host necrotic local lesions were produced similar to those described by Yarwood (8). Local lesions were also obtained on Blackeye cowpeas, snap bean (vars. Landreth, Contender, Black Valentine), tobacco, cucumber, squash, cantaloupe, corn, and oats. No systemic symptoms were observed on any host. Tests showed that the thermal inactivation point was above 80°C. In other ways the virus acted in a manner similar to that described by other workers (4, 7) for tobacco necrosis. Consistent results were obtained only during the months of November, December, January, and February in a greenhouse maintained at a cool (65° F.) temperature.

The usual methods of mechanical transmission were used. Pieces of washed roots were ground with a small amount of .1 M dibasic sodium phosphate buffer. Inoculum was rubbed gently on onto leaves dusted with carborundum. Inoculated plants were washed with tap water immediately following inoculation. Ordinarily Pinto beans were used as test plants. They were inoculated at the time the unifoliate leaves were fully expanded.

It was assumed that the virus was introduced into the greenhouse in the plants of Fragaria vesca (East Malling clone). This assumption was based on the following observations. The virus was rather constantly associated with plants of the East Malling clone or with plants that had been grafted to this clone. It was only rarely obtained from other strawberry plants growing in this greenhouse. During the winter strawberry plants were dug from several commercial strawberry fields and tobacco necrosis virus was never obtained from these plants. Since other workers (4, 5, 6) have indicated that the virus can be introduced in the potting mixture or in the water, these possibilities were considered. Field-grown strawberry plants were potted in the regular potting mixture, in sand, and in steam-sterilized sand. The plants in sand were watered with a nutrient solution and the others were watered with tap water. Parts of each lot of plants were shaded and the remainder were placed on the regular greenhouse benches. In each case precautions were taken to prevent contamination of the pot from below. Roots from these plants were used to inoculate Pinto bean plants prior to potting, after two months, and after three months. In no case was tobacco necrosis virus obtained from any of these plants.

Tobacco necrosis viruses are ordinarily believed to remain localized in plant tissues even when associated with roots. Observations with strawberry plants indicate that the virus encountered in this work readily passes on to runner plants either through plant tissue or by contamination from close association. It may be that the virus in strawberry exhibits a type of systemic development similar to that described by Bawden and Kassanis (1) with Primula obconica or by Bawden and Van der Want (2) with bean stipple-streak. The tobacco necrosis virus was obtained from strawberry leaves on only one occasion. Numerous other trials with leaf tissue failed to yield it, indicating that the single positive trial may have been due to some type of contamination.

The passage of the virus through runners or runner-grafts to the daughter plants was considered briefly. Runners from Fragaria vesca (East Malling clone) were rooted in steam-sterilized soil. In some cases runners from East Malling clone plants were grafted to runners from plants of a clone of Fragaria virginiana that had been consistently free of the tobacco necrosis virus. Precautions were taken to prevent contamination of the pots from below. After two weeks the tobacco necrosis virus was obtained from only the East Malling clone mother plants. After one month the virus was obtained from East Malling clone daughter plants and from both the mother and daughter plants of the grafted F. virginiana plants. Ungrafted F. virginiana plants set in the same area remained free of the virus. These results do not necessarily show that the virus pass-

[1]Research Paper No. 1052, Journal Series, University of Arkansas. Published with the permission of Director of the Arkansas Agricultural Experiment Station.

ed from one plant to the other only through the tissue union. They do show that the passage of the virus from plant to plant is facilitated by this close proximity.

The observations presented here are of a limited nature and show only that one of the tobacco necrosis viruses can be associated with strawberry roots. The writer has no evidence to show that the entire East Malling clone of Fragaria vesca as it exists in the United States is contaminated with this virus. Plants of this clone were obtained from another source in March. Several inoculations from the roots failed to reveal the presence of the virus. At that time of year, however, inconsistent results were obtained in other inoculations with this virus. Other workers may encounter plants of this clone or other clones contamined with a tobacco necrosis virus. It would appear to be quite inadvisable to use such contaminated plants in an indexing program. Efforts should be made to avoid distributing this virus about the country on strawberry plants wherever possible. It may also be of interest to note that this represents one of the few cases to date of ready transmission of a virus from strawberry plant tissue by mechanical means.

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AGRICULTURAL EXPERIMENT STATION, UNIVERSITY OF ARKANSAS, FAYETTEVILLE

VIRUS DISEASES OF SMALL GRAINS IN ARKANSAS, 1951-52

H. R. Rosen

An exceptionally heavy aphid infestation occurred in Arkansas in the fall of 1951 in the eastern part of the State and during the winter and spring of 1951-52 in the western part. The marked increase in aphids this spring was probably due in large measure to the prolonged mildness of the winter weather after December. What little winter weather we had came in November and in the first three weeks of December.

By February 22, the aphid infestation on all early fall-planted small grains was so heavy at the University Farm, Fayetteville, that many of the lower leaves, upon which aphid numbers were most concentrated, were dying. Incidentally, in the case of susceptible wheat varieties many of the lower leaves were infected with leaf rust (Puccinia rubigo-vera var. tritici) so that the early death of these leaves, induced by aphid infestations, reduced the amount of leaf rust inoculum and the subsequent epidemic of leaf rust that developed would probably have appeared much earlier and would have caused much more damage had it not been for the aphids.

The species of aphids involved at the University Farm were mostly the apple grain aphid, Rhopalosiphum prunifoliae, with some English grain aphid Macrosiphum granarium, according to Louise M. Russel of the Division of Insect Detection and Identification, U. S. Department of Agriculture. However, in the eastern part of the State, particularly in the rice-growing area around Stuttgart, greenbug, Toxoptera graminum, was the only species identified by the same authority.

"YELLOW DWARF"

The first conspicuous indications that something in addition to aphids was seriously hurting many plants came in early April, when most oats were beginning to shoot at the University Farm. It was noted that many individual stools and occasionally whole plants were failing to shoot, remaining in the rosette stage, and either showing very marked chlorosis, particularly toward the tips of the leaves but often involving whole blades, or with yellow streaks between the veins and along the margins, the color along the veins remaining green.

These stunted, chlorotic plants often appeared in relatively large or small spots, or in contiguous parts of drill rows, or as single plants, and at first glance appeared to be characteristic of plants suffering from nitrogen starvation or of plants growing in low wet places which also frequently exhibit symptoms of nitrogen deficiency. But within these spots plants of normal height and of normal green color were often present, and the fields at the University Farm where these oats were grown had received 20 pounds of nitrogen in the fall in the form of 400 pounds of 5-10-5 fertilizer, and about 30 pounds of additional nitrogen in late February in the form of 100 pounds of ammonium nitrate per acre. Furthermore, both applications had been made with a good distributor, and none of the dark green and light green streaks that characterize uneven distribution of fertilizer were observed across the fields. Again, up until the middle of May there had been a shortage of rainfall and there was little or no opportunity for water to stand in spots for any length of time. It seemed obvious that the stunting and chlorosis was not due to any marked shortage of nitrogen or to poor drainage.

As the season advanced many of the stunted, chlorotic plants produced panicles that were also chlorotic, the outer glumes of the individual spikelets being of a sickly yellowish color. These panicles usually showed a high percentage of sterility or blast. However, many stools failed to produce panicles and gradually waned and died. Within a week or so after their death they become covered with blackish masses of conidia and conidiophores of Helminthosporium avenae. Many of the plants that did produce panicles showed premature ripening and death, with poorly filled grains or none at all. Such plants also became covered with black moulds, largely, if not entirely, H. avenae.

The general appearance of the larger field spots where there was much chlorosis and stunting contrasted rather sharply with the appearance of spots where aphids themselves seemed to be the chief offenders. Where there was chlorosis and stunting there often was a gradual decline in height of plants from the margin to the center of the spot, whereas in the case of aphid injury there was no noticeable gradation in height of plants, although the amount of injury and death of plants was greatest at the center.

In addition to stunting and to chlorosis of the upper leaves including the flag leaf, most of the plants with such symptoms also showed a leaf reddening confined mainly to the lower leaves. However, there was much leaf reddening of plants that showed little or no stunting and no chlorosis. In some oat varieties, notably in Arkwin and Fulwin, little stunting or chlorosis appeared at least until the early milk stage, and yet there was much leaf reddening of the lower leaves. Also, in

spots in the field plants of these varieties showed an extreme amount of leaf reddening usually accompanied by a large number of leaf spots (spot blotch) induced by H. avenae. It is assumed that such field spots marked the center of heavy aphid infestations and that the large number of leaf spot infections indicated that aphid injury is conducive to such infections. (Of course, it is well known that H. avenae can infect without aphid injury).

It appears that there were three different kinds of injury in the field: first, direct aphid injury associated with a marked reddening, browning, and killing of the foliage of oats; second, a reddening of lower leaves plus a chlorosis, stunting and premature ripening of plants; and third, a red spotting or mottling. The three seemingly could be distinguished by the difference in reaction of unlike varieties. While all varieties tested showed considerable leaf reddening, there was a marked difference between varieties in the amount of chlorosis and stunting and of red spotting. The following varieties and selections showed the least amount (trace) of chlorosis and stunting: Arkwin, Fulghum 708, Fulwin, and R 22-36b-9-647, with Atlantic, Arlington, and Mustang showing 5 to 10 percent. The following varieties showed the greatest amount of chlorosis and stunting (the final percentages being estimated on May 7 in the larger varietal test plots, 1/50-acre replicated 6 times, and on May 9 in the small rod-row nursery replicated 3 times): In the larger field plots, Nortex 107 68 percent, Taggart 56 percent, De Soto 47 percent, Ferguson 922 41 percent, Victorgrain 48-93 39 percent, Fulgrain 4831 34 percent, Letoria 33 percent, Lee 5 32 percent, Stanton 4 23 percent, Traveler 15-8 24 percent, Traveler 15 percent. In the rod-row nursery Fla. 167 x Landhafer C.I. 6581 43 percent, (Bond-Fulghum) x Victorgrain Coker 51-1 C.I. 6577 37 percent, Appler x Trelle Dwarf C.I. 6579 35 percent, (Bond-Fulghum) x Victorgrain Coker 51-2 C.I. 6578 32 percent, (Red Rustproof x Victoria) x Norton C.I. 5372 32 percent, (Fulwin x Lee-Victoria) x Bonda C.I. 5875 28 percent, C.I. 4019 x (Fulghum x Victoria) C. I. 5873 20 percent, Nortex: Stoneville Sel. C.I. 5872 20 percent, and Fultex 18 percent. The data obtained in the nursery are obviously not nearly so reliable as those obtained in the larger field plots because of the relatively small number of plants and the marked fluctuations in disease prevalence. For example, Appler was read as having 33 percent in one nursery replicate of three rod rows and only 10 percent in each of two other nursery replicates, with an average of about 18 percent for the three. Since the two Red Rustproofs in the larger field plots, Nortex 107 and Ferguson 922, showed relatively high disease percentages, it seems clear that the nursery figures for Appler, Nortex, and probably other varieties are not very significant although there was in general fair agreement between nursery plantings and those in the larger plots.

So far as chlorosis, stunting, and premature ripening are concerned it may be assumed that the disease is of virus origin and is very similar to if not the same as that described by Oswald and Houston (PDR 35: 471-475, 1951; 36: 182-183, 1952) under the name of yellow dwarf ("yellow-dwarf", "barley yellow-dwarf" in Phytopathology 42: 15, 1952; and "cereal yellow-dwarf"). While these workers do not mention premature ripening as one of the distinctive, and incidentally one of the most important symptoms of this disease, there can be little doubt that the California disease of 1951 and that of Arkansas in 1952 are very similar. Aside from symptoms, it is of interest to note that the two species of aphids, the apple grain aphid and the English grain aphid, that gave the highest percentage of transmission of yellow dwarf in their inoculation tests, were the species involved in those parts of Arkansas where the disease was most prevalent.

It is important to note that Arkwin, Fulghum 708, Fulwin, and R22-36b-9-647(Tenn. 1922 x Bond-Iogold) are seemingly highly resistant to this disease although not immune from it. In these varieties under field conditions there is very little chlorosis, at least up to the early milk stage, and practically no stunting or premature ripening. However, after the early milk, it is very common to find the flag leaf and the leaf immediately below showing chlorotic symptoms, including streaking, when the lowermost leaves show little or none in these varieties.

Apparently the same disease or closely related diseases appeared also on barley, wheat, rye, and cheat (Bromus secalinus) at the University Farm. This requires verification, but, since Oswald and Houston obtained transmission from barley and wheat to barley, wheat, and oats, it may be that at least these three cereals were infected here with the same virus, and symptoms on rye and cheat were in general very similar to those on the other cereals mentioned.

Unlike oats, red pigment is produced only occasionally or not at all in affected leaves of the other hosts. However, this symptom has been observed at times on wheat, rye, and cheat, but not on barley.

On wheat the main symptoms observed in Arkansas consist of: A yellowing or chlorosis of whole blades or parts of blades extending from the tip for varying distances downward, with islands or long streaks of green between the chlorotic areas. In some varieties or selections there is mottling of the blades but no distortion of blades or heads. This mottling consists mainly of relatively small chlorotic streaks and irregular spots, averaging less than a cm. in length, occasion-

ally up to several cm., and a mm. or less in diameter, occasionally up to 4 mm. These chlorotic streaks and spots seemingly involve no collapse of tissues, at least as observed under a low-powdered binocular microscope, and appear to be level with the surrounding green tissue. While they are at times more numerous in the upper half or tip end of the blade, this does not appear to be a distinctive character and they often are as abundant in the lower half as in the upper.

Compared with oats and barley, very little stunting of wheat has been observed and only an occasional plant could be found that was markedly dwarfed. Such plants usually failed to set seed. Varieties with high percentages of diseased plants usually ripened prematurely, presumably because of the loss of chlorophyll from the leaves, but as there was comparatively little chlorosis prior to anthesis, and as there was little stunting, there did not appear to be much loss of wheat from the disease in 1952.

Estimates of percentage of diseased plants made when the varieties were in the milk stage in the varietal test plots, run cooperatively with the Agronomy Department, were as follows: Coker 47-27 85 percent, Quanah 85 percent, Chancellor 80 percent, Sanford 80 percent, Coastal 80 percent, Coker 47-23 80 percent, Denton 80 percent, Atlas 66 75 percent, Trumbull x Frondoso C.I. 12461 68 percent, R3-7-1-3-1 (Red Rock-Hope x Fultz-Hungarian) 60 percent, Clarkan 55 percent, 12659-2-11 (Frondoso x Trumbull-Hope-Hussar Sel.) 25 percent, and R3-5-7-1-3 (Red Rock-Hope x Fultz-Hungarian Sel.) 20 percent. It was not feasible to make an estimate of the amount of yellow dwarf on Hardired or Purplestraw because of the large amount of leaf rust on these varieties.

It seems that the only wheat varieties or selections in the varietal test plots that showed a high degree of tolerance or resistance were 12659-2-11 and R3-5-7-1-3. In the clipping plots seeded about a month earlier than the varietal test plots, where aphid infestations were considerably earlier and much heavier, the disease also appeared earlier but even here it does not appear to have caused much damage. On April 30, when estimates of percentage of diseased plants were made, and when many of the plants were just beginning to head, the highest estimate was 35 percent (one selection) while most of the varieties and selections were read as having 5 to 15 percent.

Time did not permit any adequate study of the amount of yellow dwarf on barley, rye, or cheat. It needs to be noted that symptoms on barley were quite like those described by Oswald and Houston. On rye and cheat the symptoms were very similar to those on wheat with very little dwarfing and not much chlorosis prior to anthesis.

The disease on cheat is badly in need of further study, not only to ascertain or confirm the identity of the disease, but to determine its possible role in perpetuating the disease in the absence of cereal crops.

RED SPOT MOSAIC

In addition to yellow dwarf there appeared to be another disease, probably of virus origin (PDR 32: 172-175, 1948; and 35: 151, 1951), which although not so abundant as yellow dwarf was still fairly common in the nursery and in the larger field plots at the University farm. This disease has been designated as red spot mosaic.

Since both yellow dwarf and red spot mosaic cause a marked reddening of oat foliage, it is exceedingly difficult to distinguish the two so far as this symptom is concerned. However, under field conditions, I have never observed chlorotic streaks on upper leaves as being a common symptom of red spot mosaic, nor have I observed so much stunting of oats in the latter disease. Furthermore, in the case of red spot mosaic, the most characteristic symptom is a reddish spotting or mottling of lowermost and intermediate leaves, decreasing on upper leaves, often making the disease appear very serious in the early spring and much less so as the season advances. These reddish spots gradually turn yellow or brown or gray, the change in color varying somewhat with variety and with weather conditions. No such spotting has been observed in what is assumed to be yellow dwarf.

Perhaps the most conclusive evidence indicating that the two diseases are different rests in the differential reaction of unlike varieties and selections. Since the variety Traveler is highly susceptible to red spot mosaic, selections have been made that promised resistance to this disease. One such selection is Traveler 15-8, which has shown a high degree of resistance or tolerance of red spot mosaic, especially in years when adjoining plots of Traveler were severely infected. This year, 1952, when red spot mosaic and supposedly yellow dwarf were both present, Traveler 15-8 showed a noticeably greater amount of the latter disease than Traveler, while Traveler showed characteristically a larger amount of red spot mosaic. Furthermore other varieties, notably Taggart, De Soto, Lee, Victorgrain, and the Red Rustproofs, which are seemingly highly resistant or tolerant to red spot mosaic, appear to be highly susceptible to yellow dwarf. The only varieties apparently resistant or highly tolerant to both are the Fulghums and their derivatives,

including Fulghum 708, Fulwin, and Arkwin.

Irrespective of the exact identity of these diseases, or whether there is one disease or a group of similar diseases, the important consideration appears to be that we are dealing with a group of highly infectious diseases capable of producing considerable damage at times and that certain varieties seem highly resistant or tolerant.

All that I have written above on oats concerns winter oats. On spring oats there were comparatively little leaf reddening, or chlorosis, or stunting in this State in 1952. However, Oswald and Houston's work in California, which appears to deal largely with spring oats, is sufficient to indicate the importance of these diseases on spring oats varieties.

In conclusion, it would be a mistake to assume that we are dealing here with new virulent diseases that may destroy our cereal crops in short order. I have known these diseases and suspected their virus origin for a number of years, and have learned to accept them in routine. Thus Arkwin yielded 88.6 bushels per acre and Traveler 80.7 bushels in the very plots where these virus diseases were so severe in 1952. While the diseases caused considerable damage in Arkansas in 1952, especially on winter oats, it is also true that aphids were probably more abundant on these crops this year in this State than at any time during the past 30-odd years.

AGRICULTURAL EXPERIMENT STATION, UNIVERSITY OF ARKANSAS, FAYETTEVILLE

RELATIVE ABUNDANCE OF WHEAT YELLOW STREAK-MOSAIC VIRUS IN LEAVES, CROWNS, AND ROOTS OF THE WHEAT PLANT[1]

H. Fellows, W. H. Sill, Jr., and H. H. McKinney[2]

An intensive search for a vector for the wheat streak-mosaic virus is in progress in several States. Atkinson (1) reported that the virus of "western" wheat mosaic is transmitted by the greenbug, Toxoptera graminum (Rond.). One of the authors, McKinney, obtained Atkinson's virus through the kindness of V. E. Wilson, then at Iowa State College. The comparative tests conducted at Beltsville, Maryland, have indicated that Atkinson was working with a strain of wheat streak-mosaic virus. As yet no confirmation of Atkinson's report is known to the authors.

Some insects and other pests feed above and others feed below the soil surface. With this in mind the leaves, crowns, and roots of both dormant and actively growing wheat plants were tested separately for the presence of the virus to obtain an approximate idea of the relative concentrations. It was thought that a low concentration or absence of the virus in one or more tissues might suggest the elimination of certain pests as probable or possible vectors.

The yellow strain, Marmor virgatum var. typicum McK. (2) was used to inoculate all source plants. Tissues were separated, ground by mortar and pestle, and extracts diluted 1 to 10 or less. Two- to three-leaf wheat plants, varieties Westar and Marquillo-Oro x Pawnee (sel. 462666), were inoculated by the carborundum-rubbing method with these extracts.

Nine trials using a total of 396 plants were made. Infection from leaves of infected field plants, both dormant and those starting active spring growth, was 100 percent in all trials even though symptoms on the dormant leaves were either absent or very diffuse. By contrast, crowns and roots from the same dormant plants gave no infection; but from the plants starting spring growth 20 and 10 percent infection from crowns and roots, respectively, was obtained. One hundred percent infection resulted from the crowns of mosaic infected greenhouse plants two months old but only 16 percent from the roots. From greenhouse plants three weeks old there was 35 percent infection from the crowns and 15 percent from the roots. Inoculations from greenhouse infected leaves always approached 100 percent. Control plants inoculated with healthy plant extracts did not develop symptoms.

In other trials at the Plant Industry Station at Beltsville, Maryland, infected leaves and roots were assayed from greenhouse grown wheat plants, variety Michigan Amber, in the "late shooting" and "late boot" stages. Thirty to 60 plants were used in each test. Extracts from the leaves resulted in 100 percent infection, but when the inoculum was taken from roots and 5 cc. of water added per gram before maceration, the infection was zero compared to 55 percent infection when undiluted root extract was used.

Natural transmission from plant to plant via the roots was also investigated. The susceptible wheat varieties mentioned previously were planted in six-inch flower pots. Before emergence, a vertical shield of roofing paper was supported across the greatest diameter of the pot so that leaf contact during growth could be eliminated. At the two-leaf stage plants on one side of the shield were inoculated. Final readings made one month after inoculation in each of three trials showed no root transmission to the other side in 32 pots containing 236 plants.

The very low virus concentrations found in the roots, the apparent absence of natural root transmission, and the typically slow movement of exclusively root feeding wheat pests would suggest that these appear to be less probable vectors for this virus than those feeding above the soil line. The high concentrations in essentially symptomless dormant leaf tissue indicate that leaf-feeding vectors might obtain ample quantities of the virus at any time after systemic invasion.

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[2]Pathologist, Division of Cereal Crops and Diseases, U. S. Dept. of Agriculture, Assistant Professor of Botany, Kansas State College, and Senior Pathologist, Division of Cereal Crops and Diseases, U. S. Dept. of Agriculture, respectively.

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DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, AND KANSAS STATE COLLEGE, MANHATTAN, KANSAS

POWDERY MILDEW OF LETTUCE REAPPEARS IN SALINAS VALLEY

W. C. Snyder, Roy Bardin, and R. G. Grogan

In the fall of 1951, powdery mildew of lettuce (Lactuca sativa L.), caused by Erysiphe cichoracearum D. C., appeared in the Salinas Valley, California, for the second time in commercial field plantings. Pryor[1] observed the disease just ten years earlier (1941) in the same area and reported then that this was the first occurrence of the disease in the field for California.

The disease is of some interest because of the suddenness and infrequency with which it has appeared and because of its potential economic importance. There is evidence to indicate that lettuce powdery mildew is an uncommon disease elsewhere in the United States also, and that in some instances the downy mildew disease of lettuce has been reported as powdery mildew.

The appearance of powdery mildew in the Salinas Valley during the fall of 1951, therefore, was as unusual as it was sudden and striking. To most growers involved, this mildew was a new disease, and because losses in yield and reduction of quality resulted, it caused some concern.

The disease was first observed during late August in a field near Gonzales, about 19 miles south of Salinas. Surveys made in the following months revealed infected fields in the Salinas and Soledad areas and in the King City area some 50 miles south of Salinas. The distribution and severity of the disease seemed to reach a peak in October, and it was present in the Valley as late as November.

Symptoms of the mildew were characteristic of the powdery mildews. Sporulation in many affected fields was profuse, coating both upper and lower leaf surfaces with a white deposit. Older, outside leaves were most heavily infected, but in fields where the crop was only half-grown, these outer leaves were important to proper sizing and head development. Necrosis accompanied the infection and caused premature death of the foliage. An affected lettuce field could be detected at a distance by its dusty, off-color appearance due both to the sporulating fungus and the yellowing and browning of diseased leaves. A striking result of the infection was the fungus odor, discernible several feet away from the fields as well as when walking through them. This odor was also perceptible at the packing sheds when lettuce from infected fields was received for trimming and packing.

Head lettuce that became diseased near the time of head maturity was not appreciably damaged since the infection was easily removable by stripping off the outer and cap leaves. Where infected early, the size after trimming was so reduced that in some cases it did not pay to harvest the heads. Two different growers reported that they were able to harvest only two-thirds of the expected yield, while still another abandoned his field without attempting harvest because of the severity of scorching that had followed the mildew.

The variety of lettuce principally affected was Great Lakes. All of the commercial plantings coming into maturity at that time were of this variety. A small experimental variety plot at the Ferry-Morse Field Station five miles south of Salinas carried infection on a range of commercial head-lettuce varieties. There was no clear-cut evidence of variety tolerance or resistance distinguishable within this plot. However, the infection was late and the experimental planting was beyond maturity when inspected.

No explanation was found for the sudden appearance of the disease. It was observed, however, that most of the affected fields were located close to the then dry Salinas River bed. One field near Gonzales examined from this standpoint showed a gradation from severe infection in the lower part of the field nearest the river bed to slight infection in the upper part. It was not determined whether this was attributable to a humidity gradient, a relationship with the inoculum source, or other factors.

A strain of powdery mildew collected by Whitaker and Pryor[2] from wild lettuce, Lactuca scariola L., growing in southern California was found by them not to infect the common varieties of cultivated lettuce. They were able, however, to breed susceptibility into the cultivated type by crossing it with wild lettuce. On the basis of their evidence, and our observations in 1951, it would seem that the mildew that has now appeared at two different times on cultivated lettuce in the Salinas Valley is of a different biologic strain from that commonly found on wild lettuce. A possible origin for this strain is mutation of the asexual stage occurring on wild lettuce, or the perithecial stage which occurs on wild lettuce[2] and on certain other hosts.

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[1] Pryor, Dean E. 1941. A unique case of powdery mildew on lettuce in the field. Plant Dis. Reptr. 25:74.
[2] Whitaker, Thomas W., and Dean E. Pryor. 1941. The inheritance of resistance to powdery mildew Erysiphe cichoracearum in lettuce. Phytopath. 31:534-540.

Although perithecia were looked for in mildewed fields of cultivated lettuce in the Salinas Valley, none were found on this host. It seems unlikely that powdery mildew would carry through the winter there in the vegetative stage on cultivated lettuce. It is perhaps the lack of favorable conditions for the carryover in commercial fields which accounts for the heretofore infrequent occurrence of this mildew in fields of cultivated lettuce in the Salinas Valley.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA AT BERKELEY AND DAVIS AND MONTEREY COUNTY AGRICULTURAL COMMISSIONERS OFFICE

ORANGE RUST, A SERIOUS MARKET DISEASE OF COLORADO SPINACH IN 1952

G. B. Ramsey, M. A. Smith and W. R. Wright

Orange rust, caused by Puccinia aristidae Tracy, on spinach grown in the vicinity of Denver, Colorado, was reported as an important market disease in 1944[1]. Subsequently the disease was more or less inconspicuous on the market, but in May 1952 it again caused serious loss in truck shipments to Chicago. Growers have reported that once infection occurs the rust spots (Fig. 1) rapidly become conspicuous and fields of spinach often become worthless within a few days.

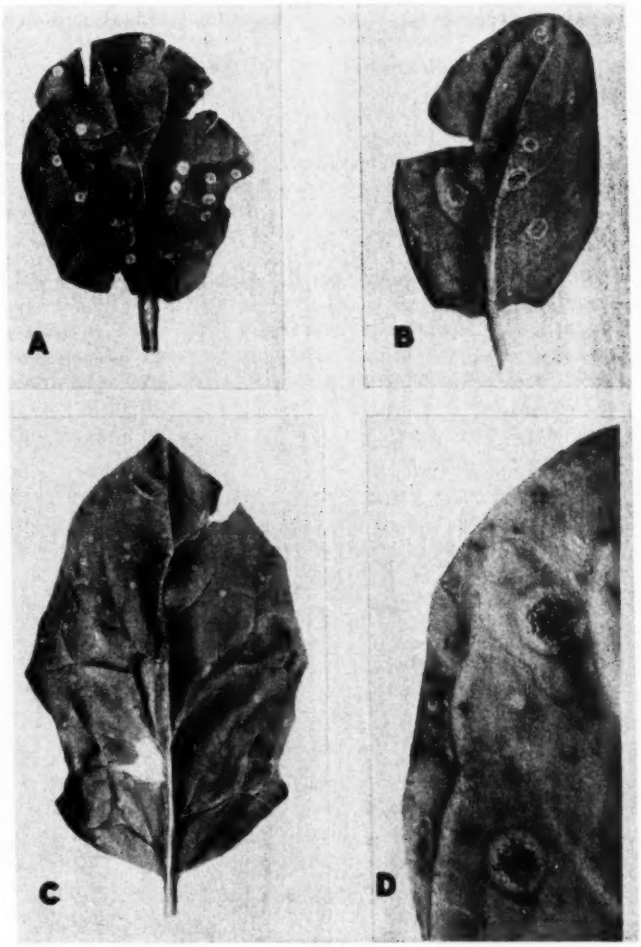

FIGURE 1. Orange rust of spinach.
A. On upper surface of curly type spinach leaf.
B. On lower surface of flat-type spinach leaf.
C. Early stages in development of sori on under side of leaf.
D. Two sori on under side of leaf, enlarged three diameters.

[1]Ramsey, G. B. and M. A. Smith. 1944. Orange rust a market factor in Colorado spinach. Plant Dis. Reptr. 28:911-912.

The first visible rust lesions on spinach are 1 mm. or less in diameter and show chiefly as light yellow spots on the under side of the leaves. Such spots are difficult for packers and shippers to recognize; consequently some infected spinach is likely to be packed unknowingly when infections occur just previous to harvest time. As the lesions enlarge and sori begin to develop the color changes rapidly from light yellow to orange and the lesions become very conspicuous on spinach in the field as well as on the market. This rapid development of rust spots on leaves in the field raised the question whether spinach with very small lesions shipped under refrigeration would be marketable after a few days in transit. A careful inspection of a truck load of spinach received in Chicago in May provides information on this question.

This load of 309 crates of spinach was shipped from near Denver in an insulated semi-trailer truck refrigerated with top ice. On arrival in Chicago on the third morning after shipment 2 to 8 inches of crushed ice remained over the top of the load and top and bottom doorway temperatures were 35° F. and 37°, respectively. These temperatures probably represent the lowest obtained in transit since it is well known that even with top ice refrigeration it takes many hours to reduce the temperature of a load of green vegetables below 45°. In various crates throughout the load 40 to 100 percent of the spinach plants showed orange rust. The average was 80 percent. The rust lesions ranged from 1 to 3 mm. in diameter, and the damage was so extensive that the entire load was unmarketable and had to be dumped.

The extremely high percentage of infection and the small size of many of the lesions suggested that a great number of infections were not apparent at loading time and had enlarged and changed color enough during transit to be readily apparent on arrival at the market. To get further evidence on the rate of development, two lots of spinach from this shipment were held for 3 days; one at 43° F. and the other at 75°. Daily examinations showed a slight increase in size and a noticeable change in color of the lesions on the spinach held at 75°. On that held at 43° there appeared to be little or no change in size of the lesions but changes in color were evident.

The results of this test and the observations of the refrigerated shipment seem to indicate that under the usual methods of handling spinach in Colorado an appreciable change in condition may be expected to occur during transit and marketing in seasons when orange rust infections are severe.

U. S. DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, CHICAGO, ILLINOIS

SAFFLOWER RUST APPEARS IN ARIZONA

J. G. Brown and Alice M. Boyle

Safflower rust, caused by Puccinia carthami Cda., has appeared in an experimental planting on the Salt River Valley Farm of the Experiment Station, Mesa, Arizona. Plants brought to us last week showed stages of the disease from pustule-spotted parts to death of the entire plant. The severely damaged plants were grown from seed of Nebraska variety No. 852 obtained via California. A nearby plot of Nebraska variety No. 8, from seed of the same source, has safflower plants apparently unaffected by the rust.

Symptoms and signs of the safflower rust, as recently described by Schuster and Christiansen[1], so far as they are based upon mature plants, are present in our specimens. There are evi-

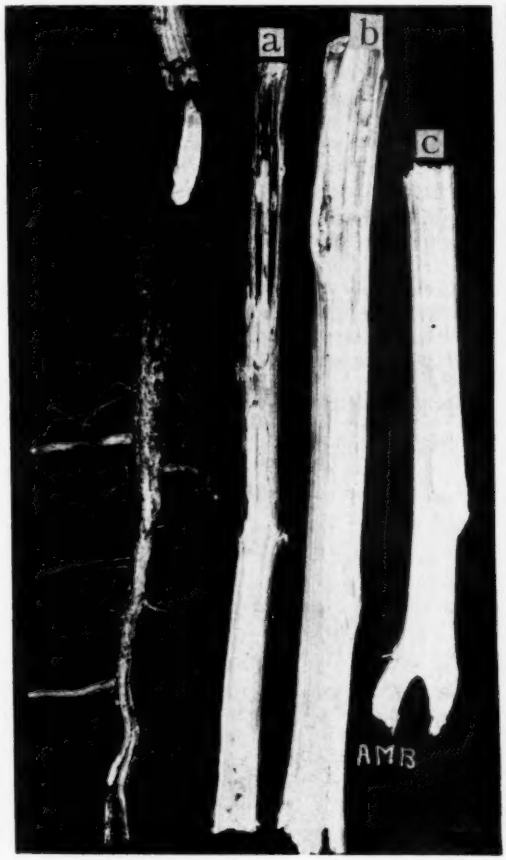

FIGURE 1. Specimens from a rust-diseased (Puccinia carthami) safflower plant grown from seed of Nebraska No. 852. Stem sections a, b, c, cut in that order from below upward, from a large sick plant. Uredo- and teleutospores present externally and internally in root and in stem sections a and b; discolored streaks in root and sections of stem.

FIGURE 2. Pustules of Puccinia carthami on leaves of safflower plant grown from seed of Nebraska No. 852.

[1]Schuster, M. L., and D. W. Christensen. 1952. A foot and root disease of safflower caused by Puccinia carthami Cda. Phytopath. 42: 211-212.

dent the same longitudinal cracking of epidermis and cortex, collapse of tissues, and internal sporulation; internal pustules contained a predominance of teleutospores over uredospores; both kinds of pustules were externally numerous on roots, stems, and leaves (Figs. 1, 2). Discolored host tissues were more pronounced basally, increasingly less so at a distance from the root upward (Fig 1).

The commercial acreage of safflower in Arizona is small, being estimated at 200 to 300 acres. No complaints of losses from rust in commercial plantings in the State have reached us. Evidently the seed used in initial plantings was clean. Later, seed of varieties of safflower described as heavier yielding was obtained and planted on the Salt River Valley Farm for testing. The rusted plants here reported were grown from this later-acquired seed.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF ARIZONA AND ARIZONA AGRICULTURAL EXPERIMENT STATION.

A PRELIMINARY REPORT OF THE COMPATIBILITY OF OAK WILT FUNGUS ISOLATES IN PENNSYLVANIA

C. L. Fergus, W. L. Yount[1], and C. L. Morris[2]

Since the discovery of oak wilt in Pennsylvania in 1950 by Fergus and Morris (4) 17 isolates of Chalara quercina Henry have been obtained from 16 different infection areas in the State. In no case have any of the single isolates or mass transfers produced fertile perithecia.

The compatibilities of the 17 Pennsylvania strains were determined in the course of crossing the strains to obtain ascospores for use in investigating ascospore germination and viability. In this study it has been found that inter-fertile strains are present in Pennsylvania. This is significant because it indicates that compatible strains exist which could possibly give rise to perithecia and ascospores. The possible importance of the role played by the ascospores in long distance above-ground spread has attracted considerable interest and speculation, although perithecia have been found only twice in nature; by Curl et al. (3) in Illinois and Staley and True (5) in West Virginia.

The compatibilities of the Pennsylvania isolates were determined by applying the conidia of one strain to the mycelial mat of another, using a sterile loop to transfer the conidia. In addition, some spermatizations were carried out by growing two different strains together on the same petri dish. Chestnut agar was the medium used in all cases. A minimum of two crosses was made in each case, but in cases in which contradictory results, or results not expected on the basis of compatibility were obtained, as many as six trials were made.

The different strains were selfed by smearing conidia produced by a strain over a chestnut agar plate culture of the same strain.

Examinations of the plates for perithecial formation were made by two methods: first, examination with the naked eye for the easily discernible globose masses of ascospores extruded from the necks of the perithecia; and secondly, examination of the cultures under a dissecting microscope at 28.7 diameters magnification. Usually, the ascospores were visible two or three

Table 1. Results[a] of spermatization attempts.

Strain number	62	55	53	67	101	64	103	128	65	127	78	115	110	1	171	174	152
62	0																
55	0	S															
53	P	P	S														
67	P	P	0	0													
101	P	P	0	0	0												
64	P	P	0	0	0	0											
103		0	P	P	0[b]	P	0										
128	P	P	0	0	0	0	P	S									
65	P	P	P[b]	0	0	0	P	0	0								
127	P	P[b]	0	0	S	0	P	0	0	0							
78	0	P	P	P			0	P	P	P	0						
115	P	P	0	0	0	0	P	0	0	0		0					
110	0	0	P	P	P	P	0	0[b]	P	P	0	P	0				
1	0	0	P	P	0[b]	P		P	P	P	0	P	S	0			
171	0		P	P	P	0	0	P	P		0	P		0	0		
174	P		0	0		0	0[b]	0	0			0		0[b]	P	0	
152	P		0	0				0	0			0			0[b]	0	0

[a] P indicates fertile perithecia produced.
S indicates sterile perithecia produced.
0 indicates no perithecial formation.

[b] Indicates a result not expected on the basis of A and B compatibility designations.

[1]Pathologist, Department of Agriculture, Commonwealth of Pennsylvania, Harrisburg, Pa.
[2]Pathologist, Division of Research, Department of Forests and Waters, Commonwealth of Pennsylvania, Harrisburg, Pa.

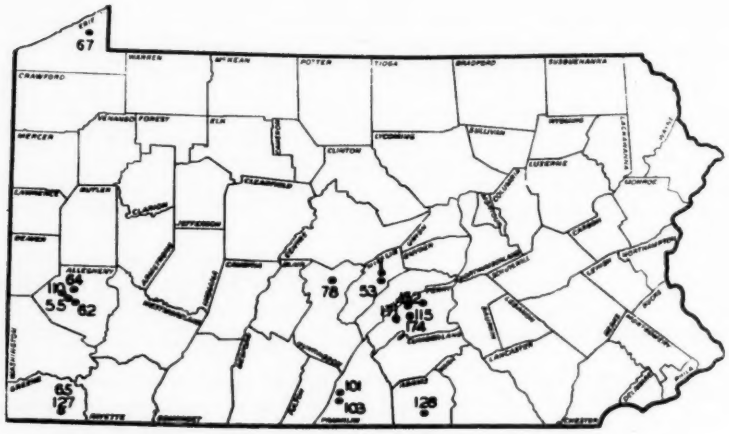

FIGURE 1. Locations of the infected trees from which the oak wilt fungus isolates used in this investigation were obtained. The numbers are the strain numbers used in Table 1.

days after spermatizing.

The results of the spermatization attempts are presented in Table 1. The map in Figure 1 shows the location of the infected trees from which the different numbered isolates were obtained.

An examination of Table 1 shows that many of the strains are inter-fertile and ascospores were produced. Of the counties in which more than one isolate of C. quercina had been made (Allegheny, Franklin, Greene, Perry, and Mifflin) inter-fertile strains were found in three: Mifflin, Allegheny, and Perry. This fact lends more strength to the conviction that as infected trees are found they should be eradicated when possible. If the infected trees are not removed perithecia could be produced by the transfer of conidia to a compatible fungus thallus in another adjacent infected tree by an insect, bird or animal. Bretz (2) has successfully used ascospores to induce the disease by artificial inoculations. Thus, ascospore production would greatly increase the amount of inoculum in an area, and in addition, might serve as the source for long distance spread.

A considerable amount of variation was observed between different strains. Differences in color of colony, texture of colony, amount of exudation of liquid, and the number of "stromatic masses" produced were observed. This variability is an interesting phenomenon and deserves further study. In fertile crosses still another variability was noted. Some crosses resulted in many more fertile perithecia than others. This was closely correlated with the number of "stromatic masses" produced by the individual strains. Cytological studies are being made to determine the exact nature of the "stromatic masses," but it seems probable that they contain the perithecial initials, or the immature perithecia. A copious exudation of liquid occurred from these masses and on removal or evaporation of the liquid (in the absence of a fertile cross) the masses were dotted with small crater-like depressions.

None of the selfed strains produced fertile perithecia; however, with three strains, perithecial necks were observed, but no masses of ascospores. When these perithecia were dissected out and examined under the compound microscope, they were found to be sterile. Bretz (1) had previously reported this phenomenon with C. quercina. These sterile perithecia with well-formed necks were observed in some non-compatible matings; however, most of the non-compatible matings did not result in their formation. Instead, the "stromatic masses" gradually dried, leaving crater-like depressions.

Isolates of known A and B compatibility designations received from Dr. G. H. Hepting and Dr. H. L. Barnett were used to determine the proper designations to apply to the Pennsylvania isolates. Strain numbers 53, 64, 65, 67, 101, 115, 127, 128, 152 and 174 are of A compatibility and strain numbers 1, 55, 62, 78, 103, 110, and 171 are of B compatibility.

The results reported here are only preliminary. Further investigations are planned for the future. The results of some of the pairings are puzzling and raise some disturbing questions. The same cross that produced perithecia in one trial did not always produce them in subsequent trials. In addition, crosses that should have resulted in fertile perithecia on the basis of compatibility did not always do so. There are two possible explanations of the confusing results. First, the technique used may not be dependable. Second, different strains of C. quercina may

vary in their ability to produce the "stromatic masses" or the perithecial initials (as was actually observed), and therefore function only as males, or as both males and females, but with a very low ability to cross with certain other strains. The writers would appreciate correspondence that might lead to a possible explanation of some of the results presented here.

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2. Bretz, T. W. 1952. The perithecial stage of Chalara quercina Henry. Phytopath. 42: 3.
3. Curl, E. A., G. J. Stessel, and B. M. Zuckerman. 1952. Macroscopic growth of the oak wilt wilt fungus in nature. Phytopath. 42: 6.
4. Fergus, C. L., and C. L. Morris. 1950. Oak wilt in Pennsylvania. Plant Dis. Reptr. 34: 291.
5. Staley, J. M., and R. P. True. 1952. The occurrence of perithecia of Chalara quercina in nature in West Virginia. Phytopath. 42: (In press).

DEPARTMENT OF BOTANY, THE PENNSYLVANIA STATE COLLEGE, STATE COLLEGE, PENNSYLVANIA

FOMES ANNOSUS ON LIVING CONIFERS

ROOTS OF LIVING PINUS RIGIDA DECAYED BY FOMES ANNOSUS

By Elmer R. Roth

Fomes annosus causes considerable root and butt rot in western hemlock, eastern white pine, and a number of European species. The fungus has also been reported as killing red cedar and Jeffrey, ponderosa, and longleaf pine, and fruiting abundantly on the stumps of many species of hard pine in the Appalachian Mountain and Piedmont regions.

Recently attention was called to the extensive rotting of the roots of two uprooted pitch pine (Pinus rigida) trees located on the flood plain of the Swannanoa River near Asheville, North Carolina. All of the primary roots were completely rotted almost up to the root collar (Fig. 1). Some of the rotted roots were more than 6 inches in diameter. The only living healthy roots were some small secondary roots that had formed near the ground level.

FIGURE 1. Decayed roots of uprooted living twin Pinus rigida tree. Note advanced typical stringy Fomes annosus decay at the breaks. Only small secondary roots (arrows) near the ground line were living.

One of the uprooted trees was about 12 inches in diameter and the other was a twin tree with stems of 14- and 16-inch diameters. In the immediate vicinity there were two standing dead trees and the stumps of two trees that had died and been cut down. In all probability these trees were killed by the root rot fungus Fomes annosus. Pure cultures from the decayed roots of the uprooted trees proved to be F. annosus.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, IN COOPERATION WITH THE SOUTHEASTERN FOREST EXPERIMENT STATION, ASHEVILLE, NORTH CAROLINA

FOMES ANNOSUS ON ATLANTIC WHITE CEDAR

By E. Richard Toole and John S. Boyce, Jr.

A recent examination of a small Atlantic white cedar (Chamaecyparis thyoides) plantation on the Bent Creek Experimental Forest near Asheville, North Carolina, showed that two-thirds of the original 50 trees were dead or dying as a result of killing and decay of roots by Fomes annosus. A few small sporophores were found at the ground line of killed trees and the fungus was readily isolated from rooted roots and stems. The plantation was established in 1927 with four-year-old seedlings from the Dismal Swamp, Virginia. The plantation was 25 years old when examined and the killed trees ranged from two to 6 inches d.b.h., and 25 to 30 feet high. Baxter[1] reported Fomes annosus on Chamaecyparis thyoides but did not indicate whether the host was living or dead.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, IN COOPERATION WITH THE SOUTHEASTERN FOREST EXPERIMENT STATION, ASHEVILLE, NORTH CAROLINA

[1] Baxter, Dow V. 1941. Some resupinate Polypores from the region of the Great Lakes. XII. Mich. Acad. Sci., Arts, and Letters Papers (1940) 26:107-121.

ADDITIONAL COLLECTIONS OF PLANTAIN SCAB AND VIOLET SCAB FROM SEVERAL NORTH CENTRAL STATES

E. A. Walker, Clark T. Rogerson, and Anna E. Jenkins

In 1949, when the senior writer was on a personal auto trip across country, he searched for scab on plantain (Plantago spp.) and on violet (Viola spp.). His collections of plantain scab, together with collections by others, are listed in Table 1. This scab has not been recorded previously in Iowa, Kansas, and Missouri. In the four States where the scab has already been recorded[1] all of the localities except Madison, Wisconsin, are new. The senior writer did not find violet scab while on his trip in 1949.

Collections of violet scab in Kansas, by Rogerson and by a violet and pansy grower in 1951, are listed in Table 2. Heretofore Texas and Louisiana were the only two States in which violet scab has been found west of the Mississippi River[2]. This is the first finding of scab on Viola patrinii DC., which is a native of Siberia.

Table 1. Collections of plantain scab in seven North Central States.

State	Locality	Date of collection	Collector	Remarks
Illinois	Camping site, across Wabash River from Vincennes, Indiana	Aug. 31, 1949	Walker	Abundant, plants much damaged
	Vicinity of O'Farrell	Sept. 1, 1949	Walker	Scant in lawn and garden
Indiana	Bedford, Court House Square	Sept. 2, 1949	Walker	Extremely abundant
Iowa	Ames, Iowa State College	Oct. 23, 1946	Deslandes[a]	Abundant on campus
		June 30, 1952	Bitancourt[a]	On campus and Agronomy Farm
Kansas	Kansas City	Aug. 31, 1949	Walker	Scant in back lawn of residence
	Topeka, lawn surrounding capitol building	Aug. 30, 1949	Walker	Very abundant
	Manhattan, Kansas State College campus	Sept. 30, 1951	Rogerson	Abundant
Missouri	Columbia	Aug. 29, 1949	Walker	Very abundant
Ohio	Cincinnati	Sept. 2, 1949	Walker	Scant along street in suburb
	McArthur, near Post Office	Sept. 3, 1949	Walker	Common along main street
Wisconsin	Madison, University of Wisconsin campus	Aug. 3, 1949	Walker	On grounds just north of the Union Building

[a] Specimen contributed to Jenkins

[1] Walker, E. A., R. A. Jehle, Anna E. Jenkins, and D. A. Preston. 1947. Additional records on distribution of plantain scab. Plant Dis. Reptr. 31: 162-166.

[2] Jenkins, A. E., and A. A. Bitancourt. 1947. Spot anthracnoses in the United States and some island possessions. Plant Dis. Reptr. 31: 114-117.

Table 2. Collections of violet scab in Kansas.

Host	Locality	Date of collection	Collector	Remarks
Viola tricolor (pansy)	Norwich	Sept. 5, 1951	Jessie Myers	Severe
Viola sp. (blue violet)	Norwich	Sept. 5, 1951	Jessie Myers	Severe
Viola patrinii	Manhattan, formal garden on Kansas State College campus	Sept. 23, 1951	C. T. Rogerson	Plants in only a small area affected. No scab was observed on native wild violets on the campus or elsewhere in Manhattan.

Plantain scab is caused by Sphaceloma plantaginis, violet scab by S. violae. When these diseases came to the second writer's attention he had had no previous experience with them. In the absence of specimens in the herbarium of the College, representative material was sent to the third writer for verification.

INSECTICIDE DIVISION, LIVESTOCK BRANCH, PRODUCTION AND MARKETING ADMINISTRATION; KANSAS STATE COLLEGE; AND DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

COLLETOTRICHUM TIP BLIGHT OF KENAF: A BRIEF HISTORY OF THE DISEASE

John T. Presley

Colletotrichum tip blight is a serious disease of kenaf, Hibiscus cannabinus, and represents one of the hazards to production at the present time. The fungus causing the disease has been identified as Colletotrichum hibisci Pollacci (1). Originally this fungus was described from Hibiscus palustris in 1894 (2). In 1927 kenaf plantings in west Java were attacked by a fungus which was identified as Colletotrichum hibisci (3). This is the earliest reference to a Colletotrichum infecting kenaf that has come to the attention of the writer.

In the late summer of 1951 a disease had become sufficiently serious in kenaf plantings in Cuba to cause concern. The disease apparently had been observed earlier, but no great significance appears to have been attached to it prior to that time. In October of 1951 apparently the same disease was observed in damaging proportions on commercial plantings in Florida. At this time the ravages of the disease, both in Cuba and Florida, were sufficient to cause concern among the growers, and pathologists directed their attention to the disease. The unusual appearance of affected plants led some investigators to suspect a virus as the cause of the disorder. This view was held both in Cuba and in Florida.

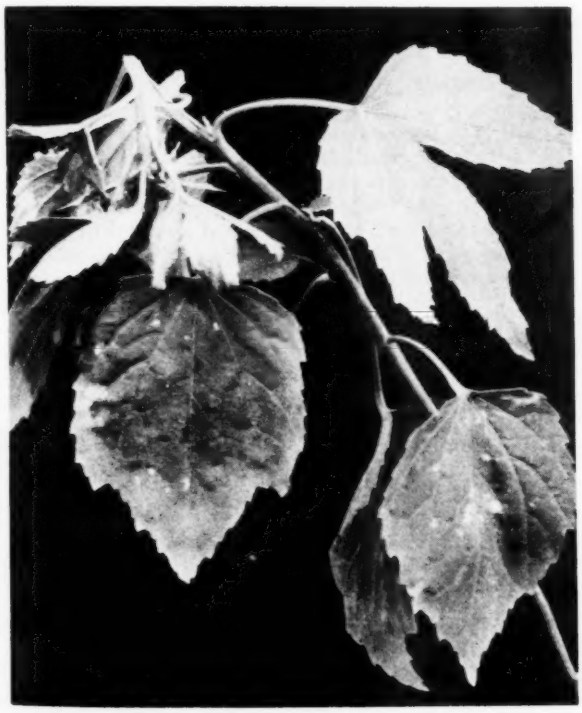


FIGURE 1. Kenaf plant four days after inoculation with Colletotrichum hibisci. Note collapse of tip and elongate lesions on upper portion of stem. Photo courtesy of O.F.A.R., U.S. Department of Agriculture.

In November 1951 specimens of diseased kenaf were received from Florida and isolations were made at Plant Industry Station, Beltsville, Maryland. A Colletotrichum was consistently recovered in these isolations and the fungus was identified as Colletotrichum hibisci (1). In January 1952 inoculations were made on kenaf plants growing in the greenhouse in Washington (4). The inoculum used consisted of a spore suspension from cultures isolated from diseased kenaf and grown in the laboratory. Four days following inoculation symptoms appeared on the kenaf plants which were typical of the early stages of the disease as observed in the field (Figure 1). When the fungus had fruited on the inoculated plants it was again identified as Colletotrichum hibisci. These results prompted the suggestion that the disease observed in specimens from Cuba and Florida was caused by Colletotrichum.

It is recognized that there may be one or more virus diseases of kenaf, but at the present writing it appears that the most serious disease and the one which has been manifest in many commercial plantings is caused by a Colletotrichum.

References and Notes:

1. Identification by John A. Stevenson, Division of Mycology and Disease Survey, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Department of Agriculture.
2. Colletotrichum hibisci, Pollacci. Atti Ist. Bot. Pavia 2 Ser. V. 1896.
3. Identification by A. Van Luijk, Phytopathology, Vol. 17, No. 1, Jan.1927, P. 26.
4. Plants for the inoculation studies were provided by Claud Horn, Office of Foreign Agricultural Relations, U. S. Department of Agriculture.

DIVISION OF COTTON AND OTHER FIBER CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, BELTSVILLE, MARYLAND

NEW OR UNUSUAL RECORDS AND OTHER BRIEF NOTES ON PLANT DISEASES

THE NORTHERN ROOT-KNOT NEMATODE, MELOIDOGYNE HAPLA, FOUND IN FLORIDA AND ALABAMA

By Vernon G. Perry

Meloidogyne hapla Chitwood 1949, sometimes commonly called the northern root-knot nematode, has recently been found infecting tomatoes and gladioli in Florida and peanuts in Alabama. This species is probably the most common root-knot nematode in the Northeastern United States but has not been reported previously from farther south than North Carolina.

In Florida M. hapla was found on tomatoes grown in a garden area in Orlando and on field-grown gladioli near Hastings. In both cases the initial infection could have been brought in on material shipped from northeastern States, since plants obtained from northern nurseries had been grown in this Orlando garden and since gladiolus corms are frequently obtained from the North.

Three peanut fields near Headland, Alabama were recently found infested with M. hapla. In one of these fields (on the Wiregrass Substation of the Alabama Experiment Station), most of the peanut plants were severely stunted during June 1952. Many of the most severely stunted plants died during an extremely hot and dry period in June.

DIVISION OF NEMATOLOGY INVESTIGATIONS

WIND DISSEMINATION OF PERONOSPORA TABACINA

By R. A. Hyre

To obtain more information on the question of wind dissemination of sporangia of Peronospora tabacina as related to the source of primary inoculum in the Maryland-Pennsylvania area, a tobacco plant bed (6 x 25 feet) was established at the University of Delaware Experimental Farm. No tobacco had been grown on the farm, or in the immediately surrounding area, for many years.

Precautions were taken to prevent contamination of the plant bed with oospores of the fungus. Tobacco seed was obtained from the Pennsylvania Southeastern Field Research Laboratory at Landisville; it was harvested from field plants grown there. As blue mold does not occur on mature tobacco in that area the possibility of oospores being introduced with the seed apparently was eliminated. Likewise, the used bed cover was autoclaved to eliminate the chance of oospores being introduced with it.

One spot of blue mold was found on June 24. (The diagnosis was verified at the Beltsville office). The weather immediately turned hot and dry and no spread of the disease was noted.

The closest plant beds in Pennsylvania were north of Homeville, 20 air miles from the University of Delaware Farm. Scattered blue mold was present in those beds. The closest plant beds in Maryland were more than 20 miles from Newark. No tobacco is grown in New Jersey within 20 miles of Newark. Four small home plantings of an ornamental species of Nicotiana have been located in Newark. None had blue mold.

It appears safe to assume that inoculum for the blue mold infection at Newark had been wind disseminated for at least 20 miles.

U. S. DEPARTMENT OF AGRICULTURE, MYCOLOGY AND DISEASE SURVEY, NEWARK, DELAWARE

VARIATION WITHIN PHYTOPHTHORA PARASITICA VAR. NICOTIANAE

By H. R. Powers, Jr. and G. B. Lucas

The causal agent of tobacco black shank, Phytophthora parasitica (Dast.) var. nicotianae (Breda de Haan) Tucker, is generally considered to be a relatively stable organism. Two lines of evidence indicate this: (1) varieties of tobacco developed for black shank resistance have maintained their resistance for many years; (2) a total of ten isolates of the black shank fungus from North Carolina, Maryland, Pennsylvania, and California, when compared to each other, showed marked similarity in regard to pathogenicity, growth rates in culture and morphological characters. However, recent observations indicate that variation does occur in P. parasitica var. nicotianae.

In April, 1952, a stock culture (originally isolated in May, 1949 from tobacco in Forsyth County, North Carolina) was used in tests for zoospore production. It was noticed that this isolate did not produce sporangia as abundantly as other isolates. Also the zoospores emerged slowly and usually in clusters. In other cultures the zoospores were released singly and within a few

seconds of one another. Sporangia from the off-type culture germinated directly by germ tubes (as opposed to indirect germination by zoospore formation) more frequently than typical cultures. Growth rates of the aberrant culture and a typical culture were equal when compared on oatmeal agar at 12°, 16°, 20°, 24°, 28° and 32° C. The aberrant culture could not be distinguished from normal cultures by gross morphological characters.

Tests were then made to compare the pathogenicity of the aberrant isolate with that of a normal isolate. Uniform disks, approximately 8.5 cm. in diameter, were cut from tobacco leaves and placed inside Petri plates that had moist filter paper fitted on both upper and lower surfaces. A small incision was made in the center of each disk and a bit of inoculum from an oatmeal agar culture was placed at this point. The normal culture produced lesions in all leaf disks within 64 hours. These lesions averaged 5.8 cm. in diameter. The aberrant culture gave variable results. In some leaf disks no lesions were produced at all. In others, lesions ranged up to 3.1 cm. in diameter, averaging 1.4 cm. Similar tests showed the abnormal isolate would not rot tomato leaves, tobacco roots, or tobacco suckers as rapidly as a normal isolate.

Further tests are being made with this weakly pathogenic strain of Phytophthora parasitica var. nicotianae and surveillance is being maintained for any further evidence of variation in this fungus.

PLANT PATHOLOGY LABORATORIES, NORTH CAROLINA STATE COLLEGE, RALEIGH, NORTH CAROLINA

BOTRYTIS ON KENAF IN NEW JERSEY

By C. M. Haenseler

In view of the recent interest in commercial production of kenaf in the United States it may be of some importance to report a severe case of Botrytis (gray mold) stem blight that occurred on an experimental planting of kenaf grown in rows 3 1/2 feet apart on the New Jersey Agricultural Experiment Station Field Crops Plots at New Brunswick in 1949. When the disease was first called to our attention the plants were 6 to 7 feet high and 1/2 to 3/4 inch in diameter. The plot was large enough and the plant population dense enough to prevent free air circulation among the plants and all environmental conditions seemed to be highly favorable for development of the disease.

No data are available on the time of initial Botrytis infection but by October 21, 1949 the disease was very prevalent and the fungus was sporulating in great abundance on practically all of the stem lesions.

Counts made on 500 consecutive canes on October 21 showed that 30 percent had one to three, or occasionally more, cankers. The individual cankers were from 1 to 15 inches long and all except the smaller ones completely encircled the stem.

The cankers were located at various heights on the stem, some near the ground and others 5 to 6 feet above the ground. A few canes had "tip blight" due to complete girdling but most of the cankers affected only the cortex and did not kill the terminal portion.

Infection usually started at a node, and apparently spread to the stem from infected petioles. It was not definitely determined whether the Botrytis first established itself as a saprophyte on dead, but still attached, petioles but this seemed to be true.

The Botrytis fruited in great abundance on the cankers, causing a gray-brown, finally dark brown, dusty covering on the entire infected area. Conidiophores were about 500 μ long and 10 to 12 μ in diameter, brown and unbranched. The spores were borne at the tip of the conidiophore and averaged 10 x 10 to 13 μ.

Seymour's Host Index lists Botrytis hortensis Ell. & Ev. on Hibiscus, but the fungus on kenaf has not been studied carefully enough to name specifically.

What may be the same species of Botrytis has been repeatedly observed in New Jersey forming cortex cankers 12 or more inches long on succulent raspberry canes. On the raspberry, as on kenaf, the cankers are largely cortical and only rarely penetrate sufficiently to kill the portion of the stem above the canker. Conidial production is much more profuse on kenaf than on raspbery and the small flat sub-epidermal sclerotia which develop on raspberry canes were not observed on kenaf.

NEW JERSEY AGRICULTURAL EXPERIMENT STATION, NEW BRUNSWICK, NEW JERSEY

INTERNAL CORK OF SWEET POTATO OBSERVED IN SOUTH FLORIDA

By Warren N. Stoner[1]

Internal cork of sweet potato described by Nusbaum[2] was found June 19, 1952 in Palm Beach and Martin Counties, Florida. Combined plantings totaling about 1500 acres near Indiantown and 105 acres near Belray Beach have developed internal cork in amounts varying from 30 to 70 percent in the saleable field-run roots. Porto Rico bunch and runner varieties were grown in all plantings.

Seed roots used for all observed fields were obtained from North Carolina, South Carolina or Georgia. Internal cork is known to occur in all of these States[3]. Certified seed roots were used for only one planting of 75 acres at Indiantown and that planting was free of the disease. Plants for most of the acreage were grown from 1951 roots culled from storage or the lowest commercial grade obtainable, but several fields were set with plants or vine cuttings taken from seedbeds which had been left intact for one to three years. This latter practice has led to a high incidence of black rot and scurf in the roots harvested.

In roots dug for internal examination from several seedbeds the incidence of internal cork was proportional to the time the seedbeds had been growing. The seedbed planted with certified roots had been partially destroyed but several roots were found and cut and all were free from internal cork. Seedbeds planted in December 1951 ran about 60 percent corky roots. Those two or three years old were almost 100 percent infected with internal cork virus. This high incidence of infection would be expected even if the original seed roots had only a trace of internal cork, since the beds had been abandoned and had no care after the first year.

A favorable market induced most growers to dig early, and at the time of observation the vines had been topped from most of the fields in preparation for harvest. Foliage symptoms of internal cork were seen, however, in two fields and one seedbed.

In one case draws were sold from a seedbed containing a high percentage of plants infected with internal cork virus for planting at Bushnell, Florida in Sumter County. This planting has not yet been examined, and consequently nothing is known of the progress of the disease in this area.

EVERGLADES EXPERIMENT STATION, BELLE GLADE, FLORIDA

[1]Assistant Plant Pathologist, Everglades Experiment Station, Belle Glade, Florida.

[2]Nusbaum, C. J. 1946. Internal cork, a new disease of sweet potato of unidentified cause. Phytopath. 36: 18-23.

[3]Nusbaum, C. J. 1950. Internal cork of sweet potatoes. South Carolina Agric. Sta. Bulletin 381.

DISTRIBUTION OF CABBAGE YELLOWS IN FLORIDA IN 1952

By A. H. Eddins

Cabbage yellows (Fusarium oxysporum f. conglutinans (Wr.) Snyder & Hansen) was first reported in Florida at Bartow in 1946[1], and in 1948 it appeared at Hastings[2]. Two new outbreaks occurred in Florida during the 1951-1952 growing season.

On December 7, 1951 diseased cabbage plants from a plant bed at Groveland in Central Florida were examined by Dr. J. M. Walter, Gulf Coast Station, Bradenton, and he concluded that it was a case of yellows. About a week later plants from the same place were examined by Dr. W. B. Tisdale, Agricultural Experiment Station, Gainesville, and by the writer at Hastings. Some of these plants also showed symptoms of yellows. Fourteen apparently healthy plants from the Groveland plant bed were transplanted to sterilized soil and kept in a greenhouse at Hastings at temperatures ranging from 60° to 80° F. Several showed symptoms of yellows a week later and by the end of four weeks all had been killed by the disease.

Diseased plants of Copenhagen Market and Bonanza from a 50-acre field near Evinston in Marion County were examined by the writer in February 1952 and found to be affected with yellows. The owner of the plants reported that it occurred scatteringly and that he had seen cabbage plants with symptoms similar to that of yellows in the crop grown in the same field in 1949.

Records show that over 1,000 acres of land in the Hastings area became severely infested with the yellows fungus during the three-year period 1948 to 1951. Losses from the disease have been reduced during the last two years, as growers have shifted from yellows-susceptible to yellows-resistant varieties for planting in infested land. Records of the leading seed dealer at

[1]Weber, George F. 1937. Two new fungous diseases of vegetables in Florida. U. S. D. A. Plant Dis. Reptr. 21: 282.

[2]Eddins, A. H., and Stanley M. Burrell. 1949. Occurrence of cabbage yellows in the Hastings Section, Florida. U. S. D. A. Plant Dis. Reptr. 33: 249-251.

Hastings show that of the 2,865 pounds of cabbage seed sold by that firm for planting in the area in 1952, 1,334 pounds or about 46 percent were of the yellows-resistant varieties Marion Market, Medium Copenhagen Resistant, Resistant Glory, and Resistant Detroit.

VEGETABLE INVESTIGATIONS LABORATORY, FLORIDA AGRICULTURAL EXPERIMENT STATION, HASTINGS

OAT DOWNY MILDEW FOUND IN FLORIDA

By R. W. Earhart

Distorted panicles of Southland oat caused by the oat downy mildew (Sclerospora macrospora Sacc.) were collected near Greenville, Florida late in May 1952. The oospores of the fungus could be distinguished in the tissues of the leaves and the glumes.

DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, AND FLORIDA AGRICULTURAL EXPERIMENT STATION, GAINESVILLE, FLORIDA

WHEAT RUST IN SOUTH DAKOTA IN LATE JUNE

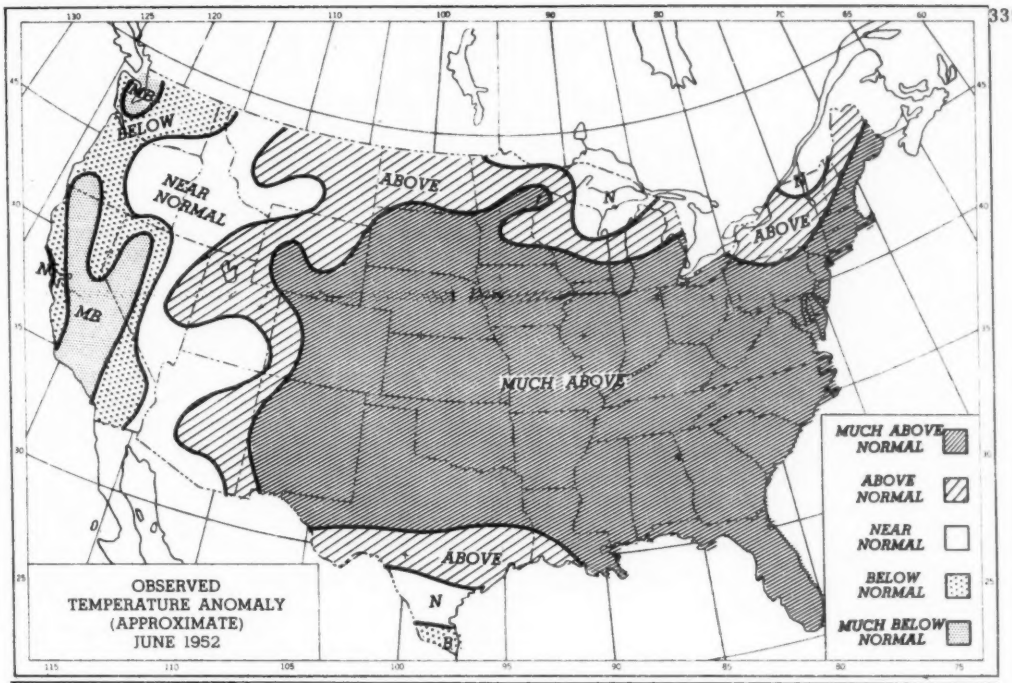
By C. M. Nagel

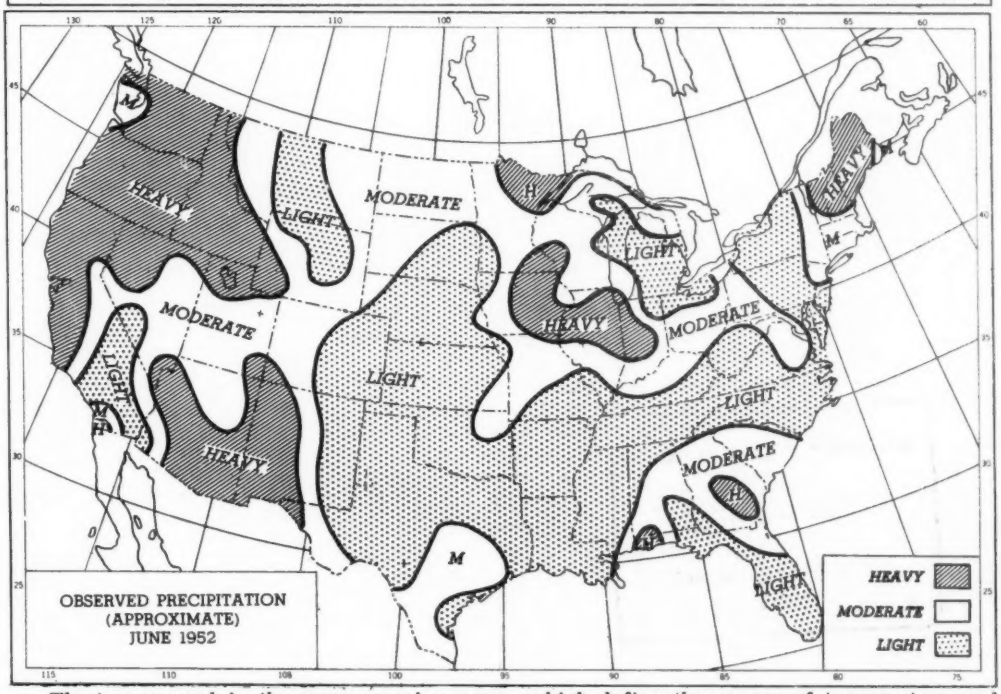
On the basis of evidence here, race 15B of stem rust, (Puccinia graminis var. tritici) is generally prevalent throughout eastern South Dakota and on the basis of prevailing weather conditions, it would appear that it could develop rapidly and cause considerable damage throughout the eastern part of the State. Also, wheat leaf rust (P. rubigo-vera var. tritici) is rapidly killing the lower leaves in certain areas. Obviously, we have had a tremendous spore shower during the past few weeks as secondary spread has not had an opportunity to take place. Currently, soil and moisture conditions are excellent in the eastern one-third of the State and it appears now that we can expect considerable damage from leaf rust and probably from stem rust as well.

SOUTH DAKOTA STATE COLLEGE OF AGRICULTURE AND MECHANIC ARTS, BROOKINGS, SOUTH DAKOTA, JUNE 25.

A CORRECTION

Our attention has been called to an error in the article by J. N. Sasser in the June 15 issue this year. On page 233 the second line in the next to last paragraph should be ".19% concentration" instead of "19%" as it reads.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

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